BRIGHAM AND WOMEN'S HOSPITAL

HARVARD MEDICAL SCHOOL

Heart & Vascular Center

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To Whom it May Concern:

Mr. Michael R Mulloy (DOB 11/7/1959) has been followed in the cardiovascular clinic at Brigham and Women's Hospital since 1999. He first presented to cardiovascular attention with onset of atrial fibrillation, and subsequently underwent cardioversion to normal sinus rhythm. He did well until 1999 when he began to note increasing symptoms of exertional fatigue/dyspnea that prompted evaluation at the University of Massachusetts. He was found to have recurrent atrial fibrillation, and was initiated on anticoagulation with warfarin and given metoprolol and digoxin for control of his heart rate. Echocardiography at that time revealed an EF of 40% without significant cavity dilation or hypertrophy. He underwent coronary angiography at that time which revealed no evidence of coronary artery disease. He was seen by Dr. James Fang at Brigham and Women's Hospital in 6/1999 who arranged an elective hospitalization for sotalol loading and repeat cardioversion. LV function subsequently recovered to normal. 3 months following cardioversion, he had evidence of recurrent atrial fibrillation, and sotalol was transitioned to metoprolol to support a strategy of rate control and anticoagulation.

Despite chronic atrial fibrillation he did well with NYHA 1 functional capacity for years, and was able to work full time in construction. Serial echocardiograms from 2001 forward continued to show normal LV ejection fraction, but did gradually reveal evidence of increasing LV wall thickness in the pattern of concentric hypertrophy with biatrial enlargement. In the absence of hypertension, this raised the question of a possible underlying hypertrophic cardiomyopathy.

In November 2011 he had an episode of unheralded syncope. This prompted a looping event recorder which noted a prolonged episode of nonsustained VT. He was referred to electrophysiology and in December 2011 underwent a implantation of a single chamber defibrillator for sudden death prevention. Since then he has had no ICD discharges, but continued to log episodes of NSVT and episodic rapid AF that prompted beta-blocker titration and addition of diltiazem with effective suppression. In November 2014 a single episode of recurrent sustained VT was terminated with antitachycardia pacing. Additional antiarrhythmic drug therapy was considered, as was an attempt at electrophysiologic study for catheter ablation, but both were deferred given the relatively infrequent and asymptomatic episodes of sustained VT.

He continued to do well until early 2015 when he experienced recurrent ICD shocks while shovelling snow. On interrogation these appeared to be inappropriate discharges related to rapid atrial fibrillation. His beta-

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blocker was uptitrated and his heart rate threshold for defibrillation was increased. Subsequent device interrogations have revealed brief episodes of polymorphic VT, but these are infrequent and he has been asymptomatic, so no therapeutic intervention has been undertaken.

He was last seen at BWH in May 2018, at which time he was doing well with NYHA Class 1 functional capacity. Last ECHO in 2/2018 revealed normal EF (64%) with concentric LVH and a calcified aortic valve with moderate aortic regurgitation (progressed only slightly in severity since 2012), but no stenosis.

It has been a pleasure to participate in his care. Should you require additional details about his cardiovascular history, please feel free to contact our office.

Sincerely,

Akshay Suvas Desai, MD, MPH